SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

This action is directed to the public in general. This action may be of particular interest to anyone who may be affected if the AEGL values are adopted by government agencies for emergency planning, prevention, or response programs, such as EPA's Risk Management Program under the Clean Air Act and Amendments Section 112r. It is possible that other Federal agencies besides EPA, as well as State agencies and private organizations, may adopt the AEGL values for their programs. As such, the Agency has not attempted to describe all the specific entities that may be affected by this action. If you have any questions regarding the applicability of this action to a particular entity, consult the DFO listed under **FOR FURTHER INFORMATION CONTACT**.

B. How Can I Get Additional Information, Including Copies of this Document or Other Related Documents?

1. *Electronically*. You may obtain electronic copies of this document, and certain other related documents that might be available electronically, from the EPA Internet Home Page at http://www.epa.gov/. To access this document, on the Home Page select ''Laws and Regulations,'' ''Regulations and Proposed Rules,'' and then look up the entry for this document under the ''**Federal Register**—Environmental Documents.'' You can also go directly to the **Federal Register** listings at http://www.epa.gov/fedrgstr/.

2. *In person*. The Agency has established an official record for this action under docket control number OPPTS–00297. The official record consists of the documents specifically referenced in this action, any public comments received during an applicable comment period, and other information related to this action, including any information claimed as Confidential Business Information (CBI). This official record includes the documents that are physically located in the docket, as well as the documents that are referenced in those documents. The public version of the official record does not include any information claimed as CBI. The public version of the official record, which includes printed, paper versions of any electronic comments submitted during an applicable comment period, is available for inspection in the TSCA Nonconfidential Information Center, North East Mall Rm. B–607, Waterside Mall, 401 M St., SW., Washington, DC. The Center is open from noon to 4 p.m., Monday through Friday, excluding legal holidays. The telephone number of the Center is (202) 260–7099.

II. Meeting Procedures

For additional information on the scheduled meeting, the agenda of the NAC/AEGL Committee, or the submission of information on chemicals to be discussed at the meeting, contact the DFO listed under **FOR FURTHER INFORMATION CONTACT**.

The meeting of the NAC/AEGL Committee will be open to the public. Oral presentations or statements by interested parties will be limited to 10 minutes. Interested parties are encouraged to contact the DFO to schedule presentations before the NAC/AEGL Committee. Since seating for outside observers may be limited, those wishing to attend the meeting as observers are also encouraged to contact the DFO at the earliest possible date to ensure adequate seating arrangements. Inquiries regarding oral presentations and the submission of written statements or chemical specific information should be directed to the DFO.

III. Future Meetings

Another meeting of the NAC/AEGL Committee is tentatively scheduled for December 2000. The exact date, location of this meeting, and chemicals to be discussed will be published in a future **Federal Register** notice.

List of Subjects

Environmental protection, Chemicals, Hazardous substances, Health.

Dated: September 15, 2000.

William H. Sanders III,

Director, Office of Pollution Prevention and Toxics.

[FR Doc. 00–24439 Filed 9–21–00; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[PF–961; FRL–6737–8]

Notice of Filing Pesticide Petitions to Establish Tolerances for Certain Pesticide Chemicals in or on Food

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the initial filing of pesticide petitions proposing the establishment of regulations for residues of certain pesticide chemicals in or on various food commodities.

DATES: Comments, identified by docket control number PF–961, must be received on or before October 23, 2000.

ADDRESSES: Comments may be submitted by mail, electronically, or in person. Please follow the detailed instructions for each method as provided in Unit I.C. of the **SUPPLEMENTARY INFORMATION**. To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–961 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: Mary L. Waller, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (703) 308–9354; e-mail address: waller.mary@epa.gov.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be affected by this action if you are an agricultural producer, food manufacturer or pesticide manufacturer. Potentially affected categories and entities may include, but are not limited to:

Categories	NAICS codes	Examples of potentially affected entities
Industry	111 112 311 32532	Crop production Animal production Food manufacturing Pesticide manufacturing

This listing is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. Other types of entities not listed in the table could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether or not this action might apply to certain entities. If you have questions regarding the applicability of this action to a particular entity, consult the person listed under **FOR FURTHER INFORMATION CONTACT**.

B. How Can I Get Additional Information, Including Copies of this Document and Other Related Documents?

1. *Electronically*. You may obtain electronic copies of this document, and

certain other related documents that might be available electronically, from the EPA Internet Home Page at http://www.epa.gov/. To access this document, on the Home Page select ''Laws and Regulations'' and then look up the entry for this document under the ''**Federal Register**—Environmental Documents.'' You can also go directly to the **Federal Register** listings at http://www.epa.gov/fedrgstr/.

2. *In person.* The Agency has established an official record for this action under docket control number PF–961. The official record consists of the documents specifically referenced in this action, any public comments received during an applicable comment period, and other information related to this action, including any information claimed as confidential business information (CBI). This official record includes the documents that are physically located in the docket, as well as the documents that are referenced in those documents. The public version of the official record does not include any information claimed as CBI. The public version of the official record, which includes printed, paper versions of any electronic comments submitted during an applicable comment period, is available for inspection in the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

C. How and to Whom Do I Submit Comments?

You may submit comments through the mail, in person, or electronically. To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–961 in the subject line on the first page of your response.

1. *By mail.* Submit your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460.

2. *In person or by courier.* Deliver your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA. The PIRIB is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

3. *Electronically.* You may submit your comments electronically by e-mail to:''opp-docket@epa.gov'', or you can submit a computer disk as described above. Do not submit any information electronically that you consider to be CBI. Avoid the use of special characters and any form of encryption. Electronic submissions will be accepted in Wordperfect 6.1/8.0 or ASCII file format. All comments in electronic form must be identified by docket control number PF–961. Electronic comments may also be filed online at many Federal Depository Libraries.

D. How Should I Handle CBI That I Want to Submit to the Agency?

Do not submit any information electronically that you consider to be CBI. You may claim information that you submit to EPA in response to this document as CBI by marking any part or all of that information as CBI. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. In addition to one complete version of the comment that includes any information claimed as CBI, a copy of the comment that does not contain the information claimed as CBI must be submitted for inclusion in the public version of the official record. Information not marked confidential will be included in the public version of the official record without prior notice. If you have any questions about CBI or the procedures for claiming CBI, please consult the person identified under **FOR FURTHER INFORMATION CONTACT**.

E. What Should I Consider as I Prepare My Comments for EPA?

You may find the following suggestions helpful for preparing your comments:

1. Explain your views as clearly as possible.

2. Describe any assumptions that you used.

3. Provide copies of any technical information and/or data you used that support your views.

4. If you estimate potential burden or costs, explain how you arrived at the estimate that you provide.

5. Provide specific examples to illustrate your concerns.

6. Make sure to submit your comments by the deadline in this notice.

7. To ensure proper receipt by EPA, be sure to identify the docket control number assigned to this action in the subject line on the first page of your response. You may also provide the name, date, and **Federal Register** citation.

II. What Action is the Agency Taking?

EPA has received a pesticide petition as follows proposing the establishment and/or amendment of regulations for residues of certain pesticide chemical in or on various food commodities under section 408 of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that this petition contains data or information regarding the elements set forth in section 408(d)(2); however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petition. Additional data may be needed before EPA rules on the petition.

List of Subjects

Environmental protection, Agricultural commodities, Feed additives, Food additives, Pesticides and pests, Reporting and recordkeeping requirements.

Dated: September 6, 2000.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

I. BASF Corporation Agricultural Products

7E4885

EPA has received a pesticide petition 7E4885 from BASF Corporation, Agricultural Products, P.O. Box 13528, Research Triangle Park, NC 27709 proposing, pursuant to section 408(d) of the FFDCA, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing tolerances for residues of epoxiconazole, (2RS,3SR)-3-(2-chlorophenyl)-2-(4-fluorophenyl)-2-(1H-1,2,4-triazol-1-yl)methyl oxirane in or on bananas at 0.5 parts per million (ppm) and in or on banana pulp at 0.2 ppm. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism.* The metabolism in bananas was investigated using 14C labeled epoxiconazole. On average 70% of the total residue could be identified as parent. This corresponds to approximately 80% of the residue extractable from the peel and

approximately 90% of the residue extractable from the edible portion, pulp. Based on this result, a parent only method was developed to analyze residues from the magnitude of the residue trials.

Under worst case practices (unbagged bananas) residue in the whole fruit ranged from <the limit of quantitation (LOQ) (0.025 milligrams/kilograms (mg/kg) to a maximum of 0.41 mg/kg. Banana pulp residues from bagged bananas ranged from < the LOQ (0.025 mg/kg) to 0.17 mg/kg and averaged 0.036 mg/kg. The average value was calculated by assuming all values below the LOQ were equal to one half the LOQ or 0.0125 mg/kg.

2. *Analytical method*. The method of analysis includes extraction, liquid/liquid partition, column clean-up quantitation by gas chromatography/electron capture detection. Forty–three whole banana samples were fortified with epoxiconazole at levels ranging from 0.025 mg/kg to 0.5 mg/kg. Recovery averaged 89.3% +/- 12.4%. Forty–one banana pulp samples were fortified with epoxiconazole at levels ranging from 0.025 mg/kg to 0.25 mg/kg. Recovery averaged 88.8% +/- 9.2.%.

3. *Magnitude of residues*. Fifteen crop residue trials were conducted in the banana growing regions of Mexico, South and Central America including three sites in Colombia, four sites in Costa Rica, four sites in Ecuador, one site in Guataemala, two sites in Hondouras, and one site in Mexico. Four sequential applications were made at the 90 g/ha, slightly higher than the maximum use rate 75 g/ha to both bagged and unbagged bananas at each site. Fruit from both the bagged and unbagged treatments were harvested at 0 days following the last application.

Whole fruit (peel and pulp) samples and pulp only samples were analyzed from all treatments at all sites. Under typical practices (bagged bananas) residue in the whole fruit ranged from < the LOQ (0.025 mg/kg) to a maximum of 0.082 mg/kg. Banana pulp residues from bagged bananas ranged from < the LOQ (0.025 mg/kg) to 0.05 mg/kg and averaged 0.013 mg/kg. The average value was calculated by assuming all values below the LOQ were equal to one half the LOQ or 0.0125 mg/kg.

B. Toxicological Profile

1. *Acute toxicity*. The acute toxicity studies place technical epoxiconazole in acute toxicity category III for acute oral, dermal, and inhalation; and in acute toxicity category IV for skin and eye irritation; and the technical material is not a skin sensitizer.

2. *Genotoxicty*. A modified Ames test (3 studies; point mutation): Negative; *E. coli* reverse mutation assay (1 study; point mutation): Negative; *in vitro* chinese hampster ovary/hypoxanthine guanine phophoribosyl transferase (CHO/HGPRT) mammalian cell mutation assay (1 study; point mutation): Negative; *in vitro* Cytogentics—CHO cells (1 study; chromosome aberrations): Negative; mouse micronucleus assay (1 study; chromosome aberrations): Negative; *in vitro* unscheduled DNA synthesis (UDS) test using rat hepatocytes (1 study; DNA damage and repair): Negative; *in vivo* DNA binding in rats and mice (1 study; DNA binding): Negative.

3. *Reproductive and developmental toxicity*. i. A developmental study via oral gavage in rabbits resulted in dosages of 0, 20, 50, and 80 mg/kg/day highest dose tested (HDT) with a developmental toxicity no observed adverse effect level (NOAEL) of 80 mg/kg/day and a maternal toxicity of 20 mg/kg/day based on the following:

a. Decreased body weight (bwt), food consumption, uterus weight, and increased resorption rate and post-implantation losses in the 80 mg/kg/day dose level.

b. Slight decreases of body weight and food consumption was seen in the 50 mg/kg/day dose level.

c. No substance-related findings were observed in any fetus at all dose levels.

ii. A developmental study was conducted via oral gavage in rats resulted in dosages of 0, 5, 15, and 45 mg/kg/day HDT with a developmental toxicity NOAEL of 5 mg/kg/day and a maternal toxicity of 5 mg/kg/day based on the following:

a. Signs of maternal toxicity, in the form of decreased body weights, food consumption, and increased placental weights observed at the highest dose tested.

b. Maternal animals in the 45 mg/kg/day showed an increase in the number of late resorptions as compared to controls.

c. Increased placental weights in the 15 mg/kg/day dose level.

d. A significant number of fetuses with skeletal variations (especially rudimentary cervical and/or accessory 14th rib(s)) in the high dose group tested were observed. However, no malformations were observed in any pups in this study.

iii. In a second developmental study in rats via dermal exposure for 6 hours/day on intact skin with dosages of 0, 100, 400, and 1,000 mg/kg/day (HDT) with a development toxicity NOAEL of 400 mg/kg/day and a maternal toxicity of 400 mg/kg/day based on increased placental weights and a slight increase in the number of fetuses with skeletal variations was observed at the highest dose tested.

iv. A combination of two multi-generation rat reproduction studies (study A dose levels were 0, 3.0, 30, and 145 mg/kg/day and study B dose levels were 0, 0.9, 2.3, and 23 mg/kg/day). Study A was discontinued after extreme systemic toxicity was observed at 145 mg/kg/day. The following discussion summarizes the results from both studies. A reproductive NOAEL of 2.3 mg/kg/day and with a parental NOAEL of 2.3 mg/kg/day were determined based on:

a. Dose levels ≥23 mg/kg/day resulted in maternal death, clinical signs, clinical chemical effects, liver effects (i.e., damage), histopathology, and limited number of pregnancy and pups with reduced body weights which increased in severity to the upper dose levels, this also indicated that doses above 23 mg/kg/day were considered to be beyond the maximum tolerated dose (MTD) for pregnant rats.

b. Questionable effects were observed in the 3.0 mg/kg/day dose level.

c. No treatment-related clinical signs, body weight changes, parameters of fertility and gestation, or macro- or histopathological changes were observed for the parental F0, F1, and F2 at dose levels equal to and below 2.3 mg/kg/day.

4. *Chronic toxicity*. i. A series of two 1–year dog studies (study A dose levels were 0, 1.6, 15, and 49 mg/kg/day for which a NOAEL was established in females, and study B dose levels were 0, 0.3, 0.6, 0.9, and 1.1 mg/kg/day to determine a NOAEL in males. The NOAEL was established as 1.1 mg/kg/day based on the following effects:

a. Mortality in the 49.0 mg/kg/day dose group with severe clinical signs and evidence of liver damage in those dogs which were sacrificed for humane reasons.

b. Hematological examinations demonstrated effects in either male or female dogs at dose levels ≥1.6 mg/kg/day.

c. Clinical chemical effects of varying types were seen in either male and female dogs at dose levels ≥15.0 mg/kg/day.

d. No effects were observed in male animals at levels of ≤1.1 or female dogs at dose levels of ≤1.6 mg/kg/day.

ii. Separate chronic feeding and oncogenicity studies in rats were performed to assess the chronic toxicity and oncogenic potential of epoxiconazole. The chronic toxicity study was conducted at dose levels of 0 and approximately 2, 8, 38, and 78 mg/

kg/day. The oncogenicity study was conducted at dose levels of 0 and approximately 2, 7, 40, and 80 mg/kg/day.

The results from the 2 studies are combined and summarized as follows:

The NOAEL was determined to be 2.0 mg/kg/day based on the following effects:

a. Decreases in body weights and food consumption were observed in both male and female rats at dose levels ≥38 mg/kg/day dose groups with a very slight progression of severity to the upper level.

b. Varying clinical chemical and hematological effects were observed in either male and/or female rats at dose levels ≥8mg/kg/day with a very slight progression of severity to the upper levels.

c. Increased absolute and relative liver weights were seen for males and/or females at dose levels ≥38 mg/kg/day.

d. Microscopic findings were observed in the liver for male and/or female rats at dose levels ≥38 mg/kg/day, in female adrenals at the highest dose test, and in the ovaries at dose levels ≥38 mg/kg/day.

e. An increased incidence of neoplasms occurred at dose levels greater than the MTD of 8 mg/kg/day in the females for the adrenals and ovaries. No increased number of neoplasms were seen in male rats due to the fact that the MTD in male rats was the HDT as opposed to the female rat which was significantly lower. Taking into account the results obtained in these studies, it is concluded that the reduction in body weight gain at 38 and 78 mg/kg/day levels met the criteria for a maximum tolerated dose. It has been determined that effects observed at the 10 mg/kg/day dose level achieved or approximated the MTD.

The effects on the ovaries were as follows:

• Decreasing aromatase enzyme activity which, is a response from converting both testosterone and adrostendione (male sex-steroids) into female sex steroids (e.g., estradiol). This action would result in decreased estradiol (i.e., estrogen and prolactin) and increased androgen levels (i.e., testosterone). As a consequence of reduced estradiol levels, measured LH and FSH concentrations are slightly altered.

• The increased incidences of neoplasms in the ovaries are considered to be the result of a continuous cell proliferation by these stimulating hormones of the pituitary-gonadal axis (LH and FSH).

The effects on the adrenals were as follows:

• Decreasing adrenal-cortical enzyme activity. This action would result in decreased adrenal hormones such as corticosterone levels. As a consequence of reduced corticosterone levels, pronounced ACTH concentrations are found.

• The increased incidences of neoplasms in the adrenals are considered to be the result of a continuous cell proliferation by these stimulating hormones of the pituitary-adrenal axis (ACTH).

For risk assessment purposes the results obtained at 38 and 78 mg/kg/day dose levels should not be used because an extrapolation to lower dose levels is not justified due to the unphysiological conditions in animals treated at dose levels near or at the MTD. Under these circumstances neoplastic and non-neoplastic mechanisms may be induced which will not occur at dose levels in which the animals are able to maintain their normal physiological homeostasis.

The increases in tumor incidence in endocrine organs due to hormonal imbalance are considered to have a threshold value, because at dose levels which do not induce cellular alterations via hormone levels in these organs, a subsequent proliferation and hence tumor formation cannot occur.

iii. An oncogenicity study in mice fed dosages of 0, 0.17, 0.81, 35.3, and 70.4 (males) or 205.4 (females) mg/kg/day with a NOAEL of 0.81 mg/kg/day for male and female mice based on the following effects:

a. Highly significant decreased body weights were observed in both male and/or female mice at the mid-high and highest dose tested.

b. Clinical sign of deteriorated state of general health were observed in high dose female mice.

c. Increased liver weights and microscopic findings were observed for male and female mice at dose the highest dose tested.

d. An increased incidence of neoplasms occurred at dose levels (70.4/205.4 mg/kg/day) greater than the MTD of 35.3 mg/kg/day in the male and female mice for the liver.

Taking into account the results obtained in this study, the following conclusions are drawn: The severe reduction in body weight and body weight gain at dose levels ≥35.3 mg/kg/day indicates that these dose levels exceeded the criteria for a MTD. It has been determined that liver tumor effects observed at the 70.4 and 205.4 mg/kg/day dose levels clearly exceeded the MTD. The liver necrosis observed in the male and female mice, further support the finding that the MTD was exceeded in the 70.4 and 205.4 mg/kg/day dose levels.

A series of mechanistic studies were performed to elucidate and define the liver promotion properties of epoxiconazole. The following conclusions can be drawn from the data:

• The material is a potent inducer of the hepatic cytocrome P–450 enzyme system, similar to the drug-phenobarbital.

• The material induced proliferation of the smooth endoplasmatic reticulum in the liver centrolobular hypertrophy and induction of phase 1 and phase 2 enzymes of the xenobiotic metabolism.

• The material was determined not to be an initiator of the carcinogenic process, but a promoter of initiated cells in the tumorgenesis as has been similarly shown with drug—phenobarbital.

As stated above, for risk assessment purposes the results obtained at 70.4 and 205.4 mg/kg/day dose levels should not be used because an extrapolation to lower dose levels is not justified due to the unphysiological conditions in animals treated at dose levels exceeding the MTD. Under these circumstances, neoplastic and non-neoplastic mechanisms may be induced which will not occur at dose levels in which the animals are able to maintain their normal physiological homeostasis.

5. *Animal metabolism*. Since there are no animal feed items associated with bananas, there is no likelihood of secondary residues in meat, milk, poultry or eggs. Therefore, data concerning metabolism in livestock is not required.

6. *Metabolite toxicology*. Residues of the parent molecule, epoxiconazole are the only residues of concern.

7. *Endocrine disruption*. A series of mechanistic studies were performed to elucidate and define the aromatase enzyme inhibition properties of epoxiconazole. The following conclusions can be drawn from the *in vivo* data: The effects on the ovaries are assessed to be the result of the following:

• Decreasing aromatase enzyme activity which is responsible for converting both testosterone and adrostendione (male sex-steroids) into female sex steroids (e.g., estradiol). This action would result in decreased estradiol (i.e., estrogen) and increased androgen. As a consequence of reduced estradiol levels, measured LH and FSH concentrations are slightly altered.

• The increased incidences of neoplasms in the ovaries are considered to be the result of a continuous cell proliferation by these stimulating

hormones of the regulating hormones of the pituitary-gonadal axis (LH and FSH).

The changes adrenals are assessed to be the result of the following:

• Decreasing adrenal-cortical enzyme activity. This action would result in decreased adrenal hormones such as corticosterone levels. As a consequence of reduced corticosterone levels, pronounce ACTH concentrations are found.

• The increased incidences of neoplasms in the adrenals are considered to be the result of a continuos cell proliferation by these stimulating hormones of the pituitary-adrenal axis ACTH.

C. Aggregate Exposure

1. *Dietary exposure*. For the purpose of assessing the potential chronic dietary exposure, BASF has estimated aggregate exposure based on theoretical maximum residue contribution (TMRC) from the tolerance of epoxiconazole in or on bananas at 0.2 ppm the maximum residue found in banana pulp. The TMRC is a ''worst case'' estimate of dietary exposure since it is assumed that 100% of all the crops for which the tolerances are established are treated and that pesticide residues are always found at tolerance levels. Based on the expected reference dose (RfD) of 0.011 mg/kg/day (from the NOAEL determined in the chronic dog study and a 100–fold safety factor) and the tolerance level residue chronic dietary exposure of the general population is less than 1% of the RfD.

i. *Food*. This is a new chemical and there are no other food uses except for the proposed use on bananas.

ii. *Drinking water*. No exposure is expected from drinking water as this is an import tolerance and no U.S. registrations are expected.

2. *Non-dietary exposure*. There are no non-occupational sources of exposure to epoxiconazole for the general population due to fact the action being requested is to establish a tolerance for import purposes only.

D. Cumulative Effects

BASF has considered the potential for cumulative effects of epoxiconazole and other substances which may have a common mechanism of toxicity. BASF is aware of other triazole fungicides but has no reliable toxicology information concerning those other materials which would allow a determination regarding similarity of toxicity mechanisms. Therefore, BASF has considered only the potential risks of epoxiconazole in its exposure assessment.

E. Safety Determination

1. *U.S. population*. Using the exposure assumptions described above, based on the completeness and the reliability of the toxicity data, BASF has estimated that aggregate exposure to epoxiconazole will utilize less than 1% of the RfD for the U.S. population. EPA generally has no concern for exposure below 100% of the RfD. Therefore, based on the completeness and reliability of the toxicity data, and the exposure assessment discussed above, BASF concludes that there is a reasonable certainty that no harm will result from aggregate exposure to residues of epoxiconazole, including all anticipated dietary exposure and all other non-occupational exposures.

2. *Infants and children*. The findings in the rat and rabbit are most likely as a result of excessive maternal toxicity, treatment of pregnant rats and rabbits with epoxiconazole induced embryotoxic effects which manifested themselves in the form of early resorptions and structural anomalies in the offspring. In both the rat and rabbit, the dose-effect relationship was rather steep and showed clear threshold levels. At dose levels below the threshold of maternal toxicity, reproductive parameters as well as the offsprings remained entirely unaffected.

This data demonstrate that the rat and rabbit are similarly sensitive to epoxiconazole. Additionally, the NOAEL of 1.1 mg/kg/day from the chronic dog study used to set the RfD is 4.5x and 72.7x lower than the maternal developmental NOAELs established in the rat and rabbit teratology studies, respectively. The developmental effects observed in either the rat or rabbit occurred only at maternally toxic doses. Therefore, no additional safety factor is needed for children.

Using the assumption stated for the general population, BASF concluded that the most sensitive child population group is that of children <1–year. Using the same RfD and the same conservative exposure assumptions employed in the dietary risk analysis for the general population, it was calculated that the exposure to this group is to be approximately 2% of the RfD for the use proposed in this document. Therefore, based on the completeness and reliability of the toxicity data, and the exposure assessment discussed above, BASF concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to residues of epoxiconazole, including all anticipated dietary exposure and all other non-occupational exposures.

F. International Tolerances

A maximum residue level has not been established by the Codex Alimentarius Commission for epoxiconazole in bananas.

II. Tomen Agro, Inc.

9E06020

EPA has received a pesticide petition 9E06020 from the TM–210 (SZX 0722) Fungicide Task Force, comprised of Tomen Agro, Inc., 100 First Street, Suite 1700, San Francisco, CA 94105, and Bayer Corporation, 8400 Hawthorn Road, Kansas City, MO 64120 proposing, pursuant to section 408(d) of the FFDCA, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of iprovalicarb: (1*S*)-2-methyl-1-[[[1-(4-methylphenyl)ethyl] amino] carbonyl] propyl] carbamic acid 1-methylethyl ester in or on the raw agricultural commodity imported grapes at 2 ppm and on the processed commodity imported raisins at 3 ppm. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of iprovalicarb was investigated in grapes, potatoes and tomatoes, and the metabolic pathway is similar in the three crops. The rate of degradation on plants is quite low, and the parent compound was always the major component, with quantitatively relevant metabolites formed only in potatoes. The metabolites observed in the potato were also observed in the rat. Therefore, iprovalicarb is the only residue of concern. Plant metabolism proceeds along three pathways:

i. Hydroxylation/glycosylation of parent at the 4–methyl group on the phenyl ring, followed by further conjugations.

ii. Cleavage of the amide group between the L-valine and p-methyl-phenethylamine moieties.

iii. Hydroxylation/glycosylation of parent at the phenyl-ring 3 position.

2. *Analytical method*. The proposed enforcement residue analytical method is an HPLC method with ultra violet (UV) detection. The limit of determination is 0.05 ppm in grapes, wine, juice and raisins, and the mean recovery is 94%. DFG multiresidue method S19 has been evaluated as an

analytical method for the determination of iprovalicarb residues in grapes and other commodities. The limit of quantitation (LOQ) of iprovalicarb in/on grapes is 0.01 ppm. Recoveries in spiked samples ranged from 79% to 119%, with the standard deviations ranging from 0.06 ppm to 0.16 ppm. DFG multiresidue method S 19 (with modified extraction) was successfully validated as an analytical method for the determination of residues in/on grapes and other commodities.

3. *Magnitude of residues*. The maximum measured residue resulting from treatment according to the proposed labels and representative viticulture practices was 1.40 ppm in grapes and 2.55 ppm in raisins. Measured residues in juice and wine were lower than the measured residues in grapes.

B. Toxicological Profile

1. *Acute toxicity*. The acute oral LD_{50} in Wistar rats is greater than 5,000 mg/kg body weight.

2. *Genotoxicty*. Iprovalicarb was non-mutagenic or non-clastogenic in six of six assays:

i. *Salmonella/microsome* test, with and without S9 mix.

ii. V79–HPRT forward mutation assay, with and without metabolic activation.

iii. CHO cell assay, with and without metabolic activation *in vitro*.

iv. *In vitro* rat primary hepatocyte unscheduled DNA synthesis UDS assay.

v. Mouse micronucleus test.

vi. ^{32}P-postlabelling assay of the uterus and urinary bladder of rats. Based upon these studies, iprovalicarb is non-mutagenic and non-genotoxic both *in vitro* and *in vivo*.

3. *Reproductive and developmental toxicity*— i. In a 2–generation reproduction study in Wistar rats receiving 0, 100, 2,000 or 20,000 ppm iprovalicarb in the diet, the parental NOAEL was 2,000 ppm based upon reduced body weight development and increased liver weight at 20,000 ppm. The reproductive toxicity NOAEL was 2,000 ppm (100 mg/kg bwt/day) based upon delayed body weight development in F1 and F2 pups during lactation, slightly reduced mean litter weight at birth and at day 28, increased relative liver weights and a reduced lactation index in F1 pups at 20,000 ppm.

ii. In a developmental toxicity study in Wistar rats, the maternal and developmental NOAEL was 1,000 mg/kg bwt/day (limit dose for study and highest dose tested (LD/HDT)).

iii. In a developmental toxicity study in Russian rabbits, the maternal and developmental NOAEL was 1,000 mg/kg bwt/day LD/HDT.

4. *Subchronic toxicity*— i. In the 13–week feeding study in Wistar rats, the doses were 0, 1,250, 5,000 and 20,000 ppm. The NOAEL was 5,000 ppm (372.7 mg/kg bwt/day in males; 561.4 mg/kg bwt/day in females) based upon reduced body weight gain, increased feed intake (females only), changed clinical chemistry parameters (including liver enzyme induction) and elevated absolute liver weights at 20,000 ppm.)

ii. In the 13–week feeding study in B6C3F1 mice, the doses were 0, 280, 1,400, 7,000, and 14,000 ppm in the diet. The NOAEL in males was 1,400 ppm (325.0 mg/kg bwt/day) based upon elevated water intake and a changed hematological parameter (MCV) at 7,000 ppm (1,724.6 mg/kg bwt/day). The NOAEL in females was 7,000 ppm (3,599.5 mg/kg bwt/day) based upon elevated water intake, changed parameter in the red blood count, and increased liver weights at 14,000 ppm (6,869.0 mg/kg bwt/day).

iii. In the 13–week feeding study in Beagle dogs, the doses were 0, 250, 2,500 and 50,000 ppm iprovalicarb in the diet (0, 9.1, 62.5 and 1,250 mg/kg bwt/day). The NOAEL was 250 ppm (9.1 mg/kg bwt/day) for males and females based upon liver effects (increased activity of alkaline phosphatase and hepatocellular hypertrophy in one animal) at 2,500 ppm.

5. *Chronic toxicity*— i. Wistar rats received 0, 500, 5,000 or 20,000 ppm iprovalicarb in the diet for 24 months. The NOAEL in females was 500 ppm (31.7 mg/kg bwt/day) based upon decreased body weights, changed clinical chemistry parameters (increased cholesterol concentration and decreased total bilirubin concentration), increased relative liver weights and histopathological findings (increased incidences of hepatocellular hypertrophy) at 5,000 ppm. The NOAEL in males was 5,000 ppm (262.5 mg/kg bwt/day) based upon decreased body weights, increased APh-activity, and slight increase of tumor incidences at 20,000 ppm. The histopathological NOAEL was 5,000 ppm (262.5 mg/kg bwt/day in males and 326.3 mg/kg bwt/day in females).

To further evaluate the results of the chronic feeding study in rats:

a. A special 2–day/13–week metabolism study was conducted in Wistar rats at 500 ppm and 20,000 ppm in the diet. Some quantitative differences (shift in diastereomer ratio in favor of S,R; relative higher amounts of p-methyl-phenethylamine, higher proportions of unchanged parent compound in feces) after administration of 20,000 ppm compared to the low dose of 500 ppm were observed.

b. Plasma concentrations were investigated in a special 12–week feeding study in HsdCpb:WU rats. The plasma concentrations of parent compound increased to a measurable level at a dose of 20,000 ppm in the diet. The concentration of parent in plasma was very low due to extensive metabolism during the first pass in the liver. At a dose of 20,000 ppm, the iprovalicarb-carboxylic acid (S,R) diastereomer increased in relation to the corresponding (S,S) diastereomer when compared to the low dose.

c. A bioavailability study was conducted in Wistar rats. Administration of thermodynamically stable and thermodynamically labile modifications of iprovalicarb to Wistar rats at concentrations of 2,000 and 20,000 ppm for 2 weeks resulted in no toxicologically relevant differences based upon the concentration of the main metabolite, iprovalicarb-carboxylic acid, in plasma. Therefore, the thermodynamically stable and thermodynamically labile modifications of iprovalicarb demonstrated no significant differences in intestinal absorption and bioavailability.

d. An *in vivo* ^{32}P–postlabelling assay of uterus and urinary bladder epithelium was conducted in female Wistar rats dosed at 10,000 or 20,000 ppm in the diet for 7 days. Iprovalicarb was determined to be inactive in the assay.

e. A liver foci test was conducted in male Bor: WISW (SPF-Cpb) rats that were dosed by oral gavage with 0 or 1,000 mg/kg iprovalicarb for 28 days, followed by a promotion treatment with phenobarbital over a period of 8 weeks. Iprovalicarb was determined to not have a tumor initiating potential.

Based upon the 24–month chronic feeding study in rats, plus the special studies, a dose of 20,000 ppm exerts a continuous stress on the xenobiotic metabolizing capacity of the liver that is not observed at lower doses. Moreover, iprovalicarb has no genotoxic potential and no tumor initiation potential. Therefore, iprovalicarb is not carcinogenic in rats.

ii. $B6C3F_1$ mice received 0, 280, 1,400, or 7,000 ppm iprovalicarb in the diet for up to 105 weeks. The NOAEL in males was 1,400 ppm (283.4 mg/kg bwt/day) based upon slightly higher food and water intake and slightly lower body weights at 7,000 ppm (1,566.8 mg/kg bwt/day). The NOAEL in females was 7,000 ppm (2,544 mg/kg bwt/day), the HDT. No oncogenic potential was observed in mice.

iii. Beagle dogs received 0, 80, 800 or 8,000 ppm iprovalicarb in the diet for 53 weeks. The NOAEL was 80 ppm (2.62

mg/kg bwt/day in males and 2.68 mg/kg bwt/day in females) based upon liver effects (increased serum activities of ALT and APh, cellular hypertrophy and periportal fatty change) at 800 ppm (24.69 mg/kg bwt/day in males and 28.10 mg/kg bwt/day in females). A follow-up study was conducted in Beagle dogs that received 0, 10, 20, 40, or 80 ppm iprovalicarb in their diet for 28 days. The NOAEL for microsomal liver enzyme induction was determined to be 20 ppm (0.77 mg/kg bwt/day). Microsomal liver enzyme induction was observed at the higher doses, and reversal of induction was observed within a 4–week recovery period in the 80 ppm dose group (2.93 mg/kg bwt/day).

6. *Animal metabolism*. Iprovalicarb is readily absorbed, and greater than 97.8% of the total radioactivity was eliminated in urine and feces within 48 hours of dosing. Iprovalicarb is extensively metabolized in the rat. The primary metabolites (>58% of the administered dose) were diastereomers of iprovalicarb-carboxylic acid. Eight minor metabolites, each representing less than 2% of the administered dose, were quantified.

7. *Metabolite toxicology*. The toxicity of p-methyl-phenethylamine, a rat, plant and soil metabolite, was investigated in 2 studies:

i. The acute oral LD_{50} in Wistar rats was determined to be in the range of 300 to 500 mg/kg bw.

ii. No mutagenic activity was observed in the *Salmonella/microsome* test. p-Methyl-phenethylamine was found at concentrations of <0.2% and has been determined to not be toxicologically significant.

8. *Endocrine disruption*. No endocrine disruption potential was observed in the 2–generation reproduction study, developmental toxicity studies, subchronic feeding studies, and chronic feeding studies.

C. Aggregate Exposure

1. *Dietary exposure*. There are no registered uses of iprovalicarb in the U.S., and no registrations or other tolerances are pending. Dietary exposure to iprovalicarb in the U.S. is limited to residues in/on imported grapes, grape juice, wine, and raisins.

i. *Food*. The anticipated residue in/on fresh grapes based upon the field studies is 0.50 ppm, and 35.71% of the fresh grapes consumed in the U.S. are imported. The anticipated residue in grape juice based upon the field and processing studies is 0.050 ppm, and 37.05% of the grape juice consumed in the U.S. is imported. The anticipated residue in wine based upon the field and processing studies is 0.32 ppm, and 17.38% of the wine consumed in the U.S. is imported. The anticipated residue in raisins based upon the field and processing studies is 0.91 ppm, and 8.165% of the raisins consumed in the U.S. are imported. Assuming 100% of the imported commodities are treated and have the average residue resulting from the maximum international use of iprovalicarb, the total anticipated residue is 0.000021 mg/kg bwt/day in the U.S. diet and 0.000056 mg/kg bwt/day for the most exposed sub-population, children 1 to 6 years old.

ii. *Drinking water*. Iprovalicarb is not registered for use in the United States. Therefore, there is no exposure to iprovalicarb through drinking water in the United States.

2. *Non-dietary exposure*. Iprovalicarb is not used in the United States. Therefore, there is no non-dietary exposure to iprovalicarb in the United States.

D. Cumulative Effects

Iprovalicarb is a member of a new class of chemistry and does not have a mode of action that is common with other registered pesticides. Therefore, there are no cumulative effects.

E. Safety Determination

1. *U.S. population*. The reference dose (RfD) is 0.03 mg/kg bwt/day. Based upon anticipated residues in imported commodities and assuming 100% of the imported commodities contain residue resulting from the proposed European use of iprovalicarb, the estimated chronic dietary margin of exposure of the U.S. population is 0.07% of the RfD. Therefore, there is a reasonable certainty of no harm to the U.S. population resulting from exposure to iprovalicarb residues in/on imported commodities.

2. *Infants and children*. The population subgroup with the maximum estimated dietary exposure is children age 1 to 6 years old. For this subgroup, and using the same assumptions as listed for the U.S. population, the estimated chronic dietary margin of exposure is 0.18% of the RfD. Therefore, there is a reasonable certainty of no harm to infants and children in the U.S. resulting from exposure to iprovalicarb residues in/on imported commodities.

F. International Tolerances

The following maximum residue levels are pending in the European Union: 2.0 mg/kg in/on grapes; 0.5 mg/kg in animal fat; 0.05 mg/kg in potatoes, animal meat, animal edible offal and eggs; and 0.01 mg/kg in milk.

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ENVIRONMENTAL PROTECTION AGENCY

[OPPTS–51952; FRL–6746–8]

Certain New Chemicals; Receipt and Status Information

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: Section 5 of the Toxic Substances Control Act (TSCA) requires any person who intends to manufacture (defined by statute to include import) a new chemical (i.e., a chemical not on the TSCA Inventory) to notify EPA and comply with the statutory provisions pertaining to the manufacture of new chemicals. Under sections 5(d)(2) and 5(d)(3) of TSCA, EPA is required to publish a notice of receipt of a premanufacture notice (PMN) or an application for a test marketing exemption (TME), and to publish periodic status reports on the chemicals under review and the receipt of notices of commencement to manufacture those chemicals. This status report, which covers the period from August 14, 2000 to August 25, 2000, consists of the PMNs and TMEs, both pending or expired, and the notices of commencement to manufacture a new chemical that the Agency has received under TSCA section 5 during this time period.

ADDRESSES: Comments may be submitted by mail, electronically, or in person. Please follow the detailed instructions for each method as provided in Unit I. of the **SUPPLEMENTARY INFORMATION**. To ensure proper receipt by EPA, it is imperative that you identify docket control number OPPTS–51952 and the specific PMN number in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: Barbara Cunningham, Director, Office of Program Management and Evaluation, Office of Pollution Prevention and Toxics (7401), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (202) 554–1404; e-mail address: TSCA-Hotline@epa.gov.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

This action is directed to the public in general. As such, the Agency has not attempted to describe the specific entities that this action may apply to. Although others may be affected, this action applies directly to the submitter of the premanufacture notices addressed